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Subacute Right Ventricular Pacemaker Lead Perforation: Often talked about in consent forms but very rarely seen

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ABSTRACT. Lead perforation is a rare complication of pacemakers and implantable cardioverter-defibrillators (ICDs). The incidence with pacemakers has been reported from 0.1% to 1% whereas with ICDs the range is from 0.6% to 5.2%. We report the case of a subacute ventricular perforation in a 93-year-old woman. The patient had a VVI Medtronic (Mounds View, MN) single active-fixation lead pacemaker implanted at an outside hospital for symptomatic bradycardia. Twenty-eight days later she presented to the same hospital with complaints of chest pain and shortness of breath. An electrocardiogram showed failure to capture, which was confirmed on pacemaker interrogation. The chest radiograph showed the pacer lead extending beyond the cardiac margins and a small left pleural effusion. She was then transferred to our hospital where computed tomography (CT) of the thorax confirmed lead extension through the right ventricular wall apex, pericardium, into the left pleural space, anterior to the lingua with substantial hemothorax present without hemopericardium. The lead was removed without complications using video-assisted thoracoscopy surgery (VATS). Lead perforation extending far beyond the cardiac margins has rarely been reported in the literature. It is a serious complication following pacemaker implantation, which could potentially lead to catastrophic outcomes. It is vitally important to properly diagnose and manage lead complications. Modern VATS techniques simplify the management and minimize morbidity.

KEYWORDS. cardiac pacemaker, cardiac ventricle, perforation.

Introduction

The first lead perforation was reported in 1969 by Barold and Center.1 The incidence of perforation has decreased over time because the leads have become less stiff, more flexible, and thinner.3 Complications from the placement of pacemaker leads occur in 3.9–9.6% of cases; however, the overall complication rate for implantable cardioverter-defibrillators (ICDs) has not been quantified.2–4 Lead perforations in particular are rare in pacemakers and ICDs. The rate of lead perforation with pacemakers has been reported to range between 0.1% and 1%2,3,5–9 whereas with ICDs the rate is reported to be 0.6–5.2%.1,5,8,10

Case description

A 93-year-old Caucasian woman with history of symptomatic bradycardia had a VVI Medtronic single active-fixation lead pacemaker placed at an outside facility. There were no unexpected events during the procedure and the device representative and the physician checked pacing parameters. Chest radiographs both postoperatively and the following day confirmed the placement of...
the lead in the right ventricular apex region. Device interrogation on postoperative day 1 did not reveal any change in pacing or sensing values. The patient herself did not complain of significant symptoms immediately and therefore was discharged to a nursing home in a stable condition.

Almost immediately after discharge, the patient began to report sharp left-sided chest pain, which was mild, intermittent, and self-resolving. No medical attention was sought and there was no post-procedure follow-up scheduled. The patient had underlying moderate to severe dementia, so obtaining an exact history about the character and timing of pain onset was not possible. By postoperative day 28, the pain had become persistent, severe, and was now associated with mild shortness of breath. At this point, the patient presented to the emergency room of the same facility. Her electrocardiogram showed normal sinus rhythm rate in the 60s, first-degree AV block, and no ischemic changes. It also showed failure to capture, which was confirmed with pacemaker interrogation. A chest radiograph (Figure 1) showed the pacer lead clearly extending well beyond the left heart border into the left lung zone and a small to moderate size left pleural effusion. An urgent transthoracic echocardiogram confirmed that the lead perforated the right ventricle and a small pericardial effusion was present. The patient was hemodynamically stable, and the pain had subsided somewhat. She was admitted to the intensive care unit and was conservatively managed overnight. The next day, she was referred to our hospital for more definitive treatment.

Upon arrival, she was hemodynamically stable with blood pressure of 130/70 mmHg and oxygen saturation of 98% on room air. Her body mass index (BMI) was 33 and there was no jugular venous distention. S1 and S2 were normal, there was a soft systolic murmur at the base and no pericardial rub. Other significant medical history included cerebral vascular accident with recurrent transient ischemic attacks and dementia. Laboratory data noted a slow decline in hemoglobin and hematocrit over the last 1 week. Because lead migration to this extent is not commonly seen, there was a dilemma about the correct method of explanting: electrophysiology laboratory versus operating room and cardiothoracic (CT) surgery. Within half an hour of arrival, both electrophysiology and CT surgery evaluated her. While they were discussing the optimal plan, a transthoracic echo was carried out to re-evaluate the effusion (since the first echo had been 24 h previously). The two-dimensional mode images showed a slight increase in the pericardial effusion and Doppler images indicated a small but persistent flow around the site of the perforation (Figure 2). After discussion, the decision was made to take the patient to the operating room. CT surgery then requested a CT scan of the chest, to delineate the exact course of the lead, so that they could plan the operation: video-assisted thoracoscopic surgery (VATS) versus thoracotomy. CT scan revealed that the lead extended through the right ventricular wall at the apex of the heart into the left pleural space anterior to the lingual. There was a substantial hemothorax present without hemopericardium (Figure 3).

Within 2 h of arrival, the patient was taken to the operating room where under VATS with adjuvant anterior minimally invasive anterior thoracotomy, the pacer wire was removed. An initial incision was placed on the lateral chest wall near the location of the wire tip as determined from the CT scan. After blunt dissection, the tip was noted to be floating freely in the pleural space. The tip was brought to the skin surface and secured (Figure 4). The thoracoscope was inserted and used to track the wire back to its exit site from the pericardium. Hematoma of the pericardium was noted but no active bleeding. The pericardium was incised at this site and a total of 500 ml of bloody fluid was removed from the pericardial and pleural space. There

Figure 1: Chest radiograph showing lead tip extending beyond the cardiac margins.

Figure 2: Four-chamber view of transthoracic echocardiogram showing right ventricular lead perforating apex and doppler showing small flow around the wire.
was no significant clot found in the pericardial space. The pacemaker pocket was incised and the pacemaker generator and lead were removed without any difficulty. A pericardial window was created and a French Blake drain was placed within the pericardial space.

Postoperatively, the patient did very well. The drain was removed on postoperative day 2 and the patient was discharged home on day 3. A pacemaker was not replaced on this patient. After evaluation by electrophysiology, it was deemed that a pacemaker was not required for her history of first-degree atrioventricular block with only complaints of fatigue in a bedbound elderly woman.

Discussion

Ventricular perforations are divided in three different categories: acute, subacute, and delayed. Acute perforations occur within 24 h of placement\(^1\) and have an incidence of 1–7%.\(^{4,12,13}\) Some authors contend that with newer pacers wires that the incidence is less than 1%.\(^{1,10,12}\) Subacute perforations occur within 5–29 days of placement,\(^4,11\) and delayed perforations occur more than 30 days after placement.\(^11\) Subacute and delayed perforations are rare and often not recognized.\(^1,4\) Rates of perforation are higher with active fixation leads.\(^6,11,14,15\) Subacute and delayed perforations are thought by some to be increasing due to the increased flexibility of leads which cause increased force per unit area on the ventricular wall.\(^1\)

Predictors of particularly late perforation include concomitant transvenous pacing, steroids within 7 days, older age, helical screws (active fixation leads), BMI less than 20, anticoagulation therapy, and female gender.\(^1,4,7,8,15\) The exact cause of perforation is not fully understood but several factors are thought to play a role, including smaller, thinner leads which exert an increased force per unit area on the ventricular wall.\(^1,5,8,10,11,16\) This smaller diameter and its increased force on the ventricular wall are thought to contribute to acute perforations. Increased lead slack after placement can lead to increased tension on the free wall and cause late perforations.\(^10,12,14\) Active fixation leads have a higher incidence of perforation due to lead thickness and over-torquing.\(^1,10,17\) In addition, the lead tip forces are complex and vary with the cardiac cycle and change overtime. The interaction of the lead tip forces and the ventricular wall when unbalanced can lead to perforation.\(^16\)

Acute complications of both kinds of wire placement can include cardiac tamponade, pneumothorax, hemothorax, pericardial and pleural effusions, and lead perforations. Late complications can include infection, superior vena cava thrombosis, failed sensing, failed pacing, and erosion of the lead or generator.\(^5,18\) The various complications can present with a spectrum of symptoms from asymptomatic to sudden death.\(^11\) If a patient presents with diaphragmatic pacing, pericardial friction rub, intermittent or failed pacing or sensing, muscle stimulation, increased pacing threshold, or the lead tip less than 3 mm from the radiolucent stripe of epicardium perforation must be suspected.\(^12,16,18,19\) Normal impedance and pacing parameters do not exclude perforation.\(^17\) When lead perforation is suspected, evaluation should include pacer interrogation, chest radiograph, computed tomography (CT) of the chest, and echocardiogram.\(^4\)

Treatment of ventricular perforation varies as to the timing of the perforation. Acutely, the lead can be repositioned and followed with serial echocardiograms to monitor resolution of the perforation and to detect deterioration that would require immediate intervention. Delayed and subacute perforation management may not be as straightforward or standardized. If there is no bleeding in the mediastinum, the lead can be left in place and another lead placed in for use. If there is bleeding within or outside of the mediastinum with risk of vascular or pulmonary damage, the lead must be extracted and 30 days after placement.\(^12\) For active fixation leads, the lead can be removed transvenously.

Figure 3: Computed tomography (CT) scan showing lead tip extending way beyond the cardiac margin. On scrolling the original CT scan, entire course of the lead was visible. Pleural effusion also noted.

Figure 4: Intra-operative image showing tip of the lead.
under visualization with transesophageal echocardiogram. On the other hand, passive fixation leads have bulky tips that can cause damage with transvenous extraction. The recommended method of removal involves cardiothoracic surgery cutting the tip and the lead body being removed transvenously.

**Conclusion**

Ideally, perforation would like to be avoided. There has been decreased incidence of perforation when the lead is placed on the septal wall or in the right ventricular outflow tract where the ventricular wall is thicker as opposed to the right ventricle free wall or the apex where the myocardium is thinner. To minimize the risk of perforation, the lead should be placed along the septal wall or in the right ventricular outflow tract whenever possible. In addition, the indication for placement should be reviewed on all patients prior to placement to avoid taking risks that are not necessary. Lead perforation extending far beyond the cardiac margins has rarely been reported in the literature. It is a serious complication following pacemaker implantation, which could potentially lead to catastrophic outcomes. It is vitally important to properly diagnose and manage lead complications. Close follow-up to evaluate postoperative pain, subtle changes in pacing and sensing values may provide clues to lead migration. However, when this rare complication occurs, the use of VATS techniques may be helpful to minimize morbidity from lead extraction previously requiring a major thoracotomy.

**References**